Postprint of Meta-Analysis on the Association Between Dietary Inflammatory Index and Upper Gastrointestinal Cancer Risk

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Abstract

Background The Dietary Inflammatory Index (DII), as a novel indicator for describing the inflammatory potential of diet, has been widely applied in research on chronic diseases.

Objective This study evaluates the association between DII and the risk of upper gastrointestinal cancers (UGIC).

Methods We systematically searched English-language databases (PubMed, Web of Science, Embase, Cochrane Library) and Chinese databases (Wanfang Database, CNKI, VIP Database) to include observational studies published between 2015 and 2022 that explored the association between dietary inflammatory scores and UGIC risk. Meta-analysis was performed using RevMan 5.4.1 software, pooling odds ratios (OR) and 95% confidence intervals (CI), with subgroup analyses conducted according to study region, tumor site, pathological type, sex, Helicobacter pylori infection status, and other factors.

Results A total of 11 case-control studies involving 9,015 participants were included. Meta-analysis results showed that in categorical DII, individuals in the highest DII category had an increased risk of UGIC compared to those in the lowest DII category (OR = 1.81, 95% CI: 1.65–1.97). Among different tumor types, esophageal cancer showed the highest risk increase (OR = 2.20, 95% CI: 1.69–2.86), followed by gastroesophageal junction cancer (OR = 2.04, 95% CI: 1.24–3.36), and gastric cancer risk was significantly increased (OR = 1.95, 95% CI: 1.42–2.67). Notably, esophageal squamous cell carcinoma risk (OR = 2.68, 95% CI: 1.74–4.13) increased more markedly than esophageal adenocarcinoma (OR = 2.59, 95% CI: 1.44–4.69). In continuous DII, each one-unit increase in DII was associated with a 53% increase in UGIC risk (OR = 1.53, 95% CI: 1.25–1.88). In subgroup analysis, the risk increase was more pronounced in females (OR = 2.61, 95% CI: 1.79–3.79) than in males (OR = 1.27, 95% CI: 0.89–1.83).

Conclusion A diet with high DII scores may increase the risk of UGIC, particularly evident in esophageal cancer and female populations.

Full Text

Preamble

Meta-Analysis of the Relationship Between Dietary Inflammatory Index and Upper Gastrointestinal Cancer Risk

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Abstract

Background: The Dietary Inflammatory Index (DII) has emerged as a novel indicator for characterizing the inflammatory potential of diet and has been widely applied in chronic disease research. **Objective:** This study evaluates the association between DII and the risk of upper gastrointestinal cancers (UGIC). **Methods:** We systematically searched English-language databases (PubMed, Web of Science, Embase, Cochrane Library) and Chinese databases (Wanfang Data, CNKI, VIP Database) to include observational studies published between 2015 and 2022 that examined the relationship between dietary inflammatory scores and UGIC risk. Meta-analysis was performed using RevMan 5.4.1 software to pool odds ratios (OR) and 95% confidence intervals (CI), with subgroup analyses conducted according to study region, tumor site, pathological type, sex, and Helicobacter pylori infection status. **Results:** Eleven case-control studies comprising 9,015 participants were included. The meta-analysis revealed that individuals in the highest DII category had an increased UGIC risk compared to those in the lowest category (OR = 1.81, 95% CI: 1.65–1.97). Among different tumor types, esophageal cancer showed the highest risk increase (OR = 2.20, 95% CI: 1.69–2.86), followed by gastroesophageal junction adenocarcinoma (OR = 2.04, 95% CI: 1.24–3.36) and gastric cancer (OR = 1.95, 95% CI: 1.42–2.67). Notably, esophageal squamous cell carcinoma risk (OR = 2.68, 95% CI: 1.74–4.13) was higher than that of esophageal adenocarcinoma (OR = 2.59, 95% CI: 1.44–4.69). In continuous DII analysis, each one-unit increase in DII was associated with a 53% increase in UGIC risk (OR = 1.53, 95% CI: 1.25–1.88).

Subgroup analysis indicated a more pronounced risk increase in females (OR = 2.61, 95% CI: 1.79–3.79) than in males (OR = 1.27, 95% CI: 0.89–1.83). **Conclusion:** A diet with higher DII scores may increase UGIC risk, particularly for esophageal cancer and among female populations.

Keywords: Dietary inflammatory index; Upper gastrointestinal cancer; Inflammatory diet; Meta-analysis

Introduction

Upper gastrointestinal cancers (UGIC), including gastric cancer and esophageal cancer, accounted for approximately 1.6 million new cases and 1.3 million deaths globally in 2018 according to GLOBOCAN data. Gastric cancer ranks as the fifth most common cancer worldwide, while esophageal cancer is the seventh, with both among the top ten causes of cancer-related mortality. Established risk factors for gastric and esophageal cancers include metabolic, environmental, epigenetic, and genomic factors, as well as Helicobacter pylori (Hp) or EB virus infection, nutritional status, physical activity, and lifestyle patterns. Accumulating evidence demonstrates that chronic inflammation plays a crucial role in tumorigenesis and progression, while dietary components can generate bioactive substances that promote chronic inflammation, thereby maintaining an inflammatory tumor microenvironment that facilitates cancer cell survival, proliferation, and metastasis. For instance, saturated fatty acids, omega-6 fatty acids, processed meats, and red meat contribute to the development of chronic low-grade intestinal and systemic inflammation, whereas folate, omega-3 polyunsaturated fatty acids, and fiber can reduce inflammatory factor infiltration and alleviate inflammatory responses. A meta-analysis on meat consumption reported that each 100 g/day increase in red meat intake and each 50 g/day increase in processed meat intake elevated gastric cancer risk by 26% (95% CI: 1.11–1.42) and 72% (95% CI: 0.64–1.15), respectively. Conversely, the Mediterranean diet is widely recognized as a healthy dietary pattern due to its anti-inflammatory potential.

In 2009, researchers at the University of South Carolina first proposed the Dietary Inflammatory Index (DII) to assess the potential inflammatory effects of individual diets. In 2014, Shivappa et al. reviewed nearly 1,943 articles and updated the DII scoring system based on the ability of foods, nutrients, and other bioactive compounds to modify specific serum inflammatory markers: C-reactive protein (CRP), tumor necrosis factor-α (TNF-α), interleukin-1β (IL-1β), interleukin-10 (IL-10), interleukin-4 (IL-4), and interleukin-6 (IL-6). Using representative data from regional dietary surveys across 11 countries as the standard for comparing 45 dietary components, they classified 36 as anti-inflammatory and 9 as pro-inflammatory. For each individual dietary component, exposure Z-scores were calculated relative to the "standard global dataset mean."To minimize the impact of right skewness, Z-scores were converted to cen-

tered percentiles (n) and multiplied by the corresponding inflammatory score (b) for each food component to obtain component-specific DII scores. The overall DII score was obtained by summing all component-specific scores:

$$\mathrm{DII} = b_1 \times n_1 + b_2 \times n_2 + \cdots + b_{45} \times n_{45}$$

Thus, DII enables quantitative comparison of the inflammatory potential of individual diets, with higher scores indicating more pro-inflammatory diets, holding significant epidemiological value for predicting chronic diseases associated with inflammatory dietary patterns.

DII assesses the inflammatory potential of overall dietary patterns using food frequency questionnaires (FFQ) to quantify macro- and micronutrient intake, and has been widely applied in studies examining cancer risk associations, including gastric and esophageal cancers. However, due to variations in study design, geography, population characteristics, and tumor subtypes, consistent conclusions have not been reached. Therefore, this meta-analysis was conducted to evaluate the relationship between DII scores and UGIC risk, with subgroup analyses by tumor site, pathological type, Hp infection status, region, and sex to comprehensively explore sources of heterogeneity and provide evidence-based dietary recommendations for gastric and esophageal cancer prevention.

Methods

1.1 Literature Search

We systematically searched PubMed, Web of Science, Cochrane Library, Embase, CNKI, Wanfang Data, and VIP Database from inception to October 10, 2022. Two investigators independently conducted the search using a combination of MeSH terms and free-text keywords. Search terms included "dietary inflammatory index," "DII," "anti-inflammatory diet," "upper gastrointestinal cancer," "esophageal neoplasm," "gastric neoplasm," "ESCC," "EAC," and their Chinese equivalents, as well as synonyms. Additionally, we performed manual searches of reference lists from retrieved articles and reviews to identify additional eligible studies.

1.2 Inclusion and Exclusion Criteria

Inclusion criteria: (1) Observational studies, including case-control or cohort studies; (2) Cases confirmed as upper gastrointestinal malignancies (gastric or esophageal cancer) through medical records and histopathological reports; (3) Inclusion of categorical DII metrics (highest vs. lowest DII categories); (4) Clear reporting of outcome measures such as odds ratio (OR), relative risk (RR), or hazard ratio (HR).

Exclusion criteria: (1) Non-observational studies such as reviews, expert commentaries, or case reports; (2) Unavailable full text; (3) Duplicate publications; (4) Non-Chinese or non-English literature; (5) Unreported or non-extractable effect measures.

1.3 Literature Screening and Data Extraction

Two researchers independently screened literature, extracted data, and cross-checked results. The screening scope included all potentially eligible studies identified through database searches and reference lists. During initial screening, titles and abstracts were reviewed to remove duplicates and irrelevant studies. Full texts were then thoroughly evaluated against inclusion and exclusion criteria. For studies with questionable or missing data, corresponding authors were contacted to obtain complete information. Disagreements were resolved through discussion with a third investigator. Extracted data included: (1) Study characteristics (title, first author, design, sample size, publication year, region, dietary assessment tool, number of DII components, and covariate adjustments such as total energy intake, age, sex, education, smoking, alcohol consumption, physical activity, gastroesophageal reflux, and Hp infection); (2) Baseline participant characteristics (age, sex distribution, tumor subtypes); (3) Effect measures (OR, RR, HR, and 95% CI).

1.4 Quality Assessment

Study quality was evaluated using the Newcastle-Ottawa Quality Assessment Scale (NOS), which comprises three domains: selection, comparability, and outcome measurement. Studies were classified as low quality ($ 3*points*), *moderatequality*(4–6*points*), *orhighquality*($7 points).

1.5 Statistical Analysis

Meta-analysis was performed using RevMan 5.4.1 software provided by the Cochrane Collaboration. OR and 95% CI were used as effect measures for all studies. Heterogeneity was assessed using the chi-square test with $\alpha = 0.10$. A fixed-effects model was applied when $I^2 \leq 50\%$ and $P > 0.10$ (indicating low heterogeneity), while a random-effects model was used when $I^2 > 50\%$ and $P \leq 0.10$ (indicating high heterogeneity). Subgroup analyses were conducted by cancer site, study region, age, number of DII components, and adjustment for total energy intake to explore sources of heterogeneity. Publication bias was visually assessed using funnel plots, and sensitivity analysis was performed by sequentially excluding each study to evaluate the stability of results and the potential influence of individual studies on the pooled effect size.

Results

2.1 Literature Search Process

The initial search yielded 854 articles: 387 from PubMed, 346 from Web of Science, 18 from Embase, 64 from Cochrane Library, 28 from Wanfang Data, 5 from CNKI, 3 from VIP Database, and 3 from manual searches. After removing 498 duplicates, 321 articles were excluded based on titles and abstracts (reviews, systematic reviews, conference abstracts, interventional studies, or irrelevant research). Thirty-five articles underwent full-text review, and 24 were subsequently excluded due to incomplete outcome measures or other reasons, leaving 11 high-quality English-language studies for final inclusion. The screening process is illustrated in Figure 1 [Figure 1: see original paper].

2.2 Characteristics of Included Studies

The 11 case-control studies included 3,124 cases and 5,927 controls, published between 2015 and 2021. All studies calculated DII using the 2014 Shivappa updated scoring system. All reported categorical DII, while five studies also reported continuous DII. Five studies focused on gastric cancer, and six on esophageal cancer. Among esophageal cancer studies, five reported esophageal squamous cell carcinoma and two reported esophageal adenocarcinoma. Seven studies were conducted in Asia and four in Europe. Three studies performed sex-stratified analysis, and three performed Hp infection-stratified analysis. Eight studies adjusted for total energy intake in their covariate analyses. Quality assessment using the NOS scale and baseline characteristics of included studies are presented in Table 1 .

Table 1 Baseline Characteristics and NOS Quality Evaluation of Included Studies

Study	Country	Cancer Type	FFQ Items	Sample Size (Case/Control)	DII Components	OR (95% CI)	Covariate Adjustments
Shivappa 2016[20]	Italy	Gastric	78(31)	230/143 vs 547/286	106(35)	Categorical: 2.35 (1.32–4.20); Continuous: 1.19 (1.06–1.34)	

Study	Country	Cancer Type	FFQ Items	Sample Size (Case/Control)	DII Components	OR (95% CI)	Covariate Adjustments
Lee 2017[21]	Korea	Gastric	1164	388/249 vs 776/498	106(35)	Categorical: 1.63 (1.15–2.29)	
Vahid 2018[24]	Iran	Gastric	177	82/37 vs 95/43	168(31)	Categorical: 3.39 (1.59–7.22); Continuous: 2.65 (1.73–4.07)	
Kim 2020[25]	Korea	Gastric	1125	373/242 vs 752/487	106(35)	Categorical: 1.41 (1.00–2.06)	
Ahmad 2021[26]	Iran	Gastric	270	90/66 vs 180/131	103(29)	Categorical: 3.59 (1.16–11.02)	
Shivappa 2015[18]	Italy	Esophageal	1047	304/275 vs 743/593	78(31)	Categorical: 2.47 (1.40–4.36); Continuous: 1.23 (1.10–1.38)	
Shivappa 2015[17]	Iran	Esophageal	113	47/18 vs 96/38	125(27)	Categorical: 8.24 (2.03–33.47); Continuous: 3.58 (1.76–7.26)	

Study	Country	Cancer Type	FFQ Items	Sample Size (Case/Control)	DII Components	OR (95% CI)	Covariate Adjustments
Lu 2016[19]	Sweden	Esophageal	100	594/488 vs 806/667	63(36)	Categorical: 2.42 (1.57-3.73)	
Shivappa 2017[22]	Ireland	Esophageal	80	224/189 vs 256/216	101(25)	Categorical: 1.96 (1.11-3.47)	
Abe 2018[15]	Japan	Esophageal	129	433/376 vs 1296/1122	47(19)	Categorical: 1.71 (1.54-1.90)	
Tang 2018[23]	China	Esophageal	39	359/260 vs 380/269	137(23)	Categorical: 2.55 (1.61-4.06)	

Note: DII = Dietary Inflammatory Index; FFQ = Food Frequency Questionnaire; NA = data not available. Covariate adjustments: total energy intake, sex, age, education level, interview year, smoking, alcohol consumption, BMI, physical activity, first-degree family history of cancer, Hp infection, gastroesophageal reflux, non-steroidal drug use.

2.3 Meta-Analysis Results

2.3.1 Association Between DII and UGIC Risk A significant positive association was observed between DII and UGIC risk. As shown in Figure 2 [Figure 2: see original paper], for categorical DII, individuals in the highest DII category had an increased UGIC risk compared to those in the lowest category (OR = 1.81, 95% CI: 1.65–1.97, $P < 0.00001$), with no substantial heterogeneity across studies ($I^2 = 43\%$, $P = 0.06$), warranting use of a fixed-effects model. For continuous DII, each one-unit increase was associated with a 53% increase in UGIC risk (OR = 1.53, 95% CI: 1.25–1.88, $P < 0.0001$), with significant heterogeneity observed ($I^2 = 87\%$, $P < 0.00001$), requiring a random-effects model (Figure 3 [Figure 3: see original paper]).

2.3.2 Association Between DII and Gastric Cancer Risk As illustrated in Figure 4 [Figure 4: see original paper], DII was positively associated with gastric cancer risk. Compared to the lowest DII category, the highest category showed a 95% increase in gastric cancer risk (OR = 1.95, 95% CI: 1.42–2.67). Only Lee et al.[21] performed stratified analysis by intestinal and diffuse

gastric cancer subtypes, precluding further subgroup analysis by pathological classification.

2.3.3 Association Between DII and Esophageal Cancer/GEJ Cancer Risk As shown in Figure 4, DII was positively associated with esophageal cancer risk, with the highest DII category showing a 2.2-fold increase compared to the lowest category (OR = 2.20, 95% CI: 1.69–2.86). Gastroesophageal junction adenocarcinoma risk was also elevated (OR = 2.04, 95% CI: 1.24–3.36). Risk varied by histological subtype, with esophageal squamous cell carcinoma (OR = 2.68, 95% CI: 1.74–4.13, I^2 = 74%) showing a greater increase than esophageal adenocarcinoma (OR = 2.59, 95% CI: 1.44–4.69, I^2 = 47%) (Figure 5 [Figure 5: see original paper]).

2.3.4 Subgroup Analysis Subgroup analyses were performed by study region, tumor site, pathological type, Hp infection status, and number of DII components. None of these factors were identified as sources of heterogeneity (Table 2). Regional subgroup analysis revealed a higher UGIC risk increase in European populations (OR = 2.31, 95% CI: 1.78–3.00) compared to Asian populations (OR = 1.98, 95% CI: 1.55–2.53). However, due to the lack of data from the Americas, these findings require further confirmation. Sex, FFQ administration method, and total energy intake adjustment may be potential sources of heterogeneity, but the small sample sizes in each stratum limit definitive interpretation.

Table 2 Subgroup Meta-Analysis Results for DII and UGIC Risk

Subgroup	OR (95% CI)	Heterogeneity Test I^2 (%)	Between-group Heterogeneity
Study Region			
Asia	1.98 (1.55, 2.53)	43	
Europe	2.31 (1.78, 3.00)	47	
Sex			
Male	1.27 (0.89, 1.83)	0	
Female	2.61 (1.79, 3.79)	68	
Tumor Site			
Gastric cancer	1.95 (1.42, 2.67)	0	
Esophageal cancer	2.20 (1.69, 2.86)	58	

Subgroup	OR (95% CI)	Heterogeneity Test I^2 (%)	Between-group Heterogeneity
GEJ adenocarcinoma	2.04 (1.24, 3.36)	0	
Pathological Type			
Esophageal squamous cell carcinoma	2.68 (1.74, 4.13)	74	
Esophageal adenocarcinoma	2.59 (1.44, 4.69)	47	
DII Category			
Categorical DII	1.81 (1.65, 1.97)	43	
Continuous DII	1.53 (1.25, 1.88)	87	
Hp Infection Status			
Positive	1.47 (1.08, 1.99)	0	
Negative	1.90 (1.33, 2.71)	0	
FFQ Administration			
Self-administered	1.68 (1.53, 1.85)	0	
Interviewer-administered	2.95 (1.96, 4.43)	68	
Number of DII Components			

Subgroup	OR (95% CI)	Heterogeneity Test I^2 (%)	Between-group Heterogeneity
<30	2.41 (1.88, 3.08)	0	
$ $30	2.01 (1.57, 2.57)	58	
Total Energy Intake Adjust-ment			
Yes	2.25 (1.58, 3.22)	68	
No	2.23 (1.85, 2.68)	0	
Overall	1.70 (1.53, 1.88)	<0.0001	

Note: DII = Dietary Inflammatory Index; FFQ = Food Frequency Questionnaire; Hp = Helicobacter pylori.

2.4 Sensitivity Analysis and Publication Bias

Sensitivity analysis was conducted by sequentially removing individual studies to assess their impact on the overall UGIC risk estimate and heterogeneity. The results showed minimal fluctuation in OR values after removing any single study, indicating robust and stable findings. Funnel plot assessment suggested potential publication bias among included studies (Figure 6 [Figure 6: see original paper]).

Discussion

Inflammatory components participate in the formation of the tumor microenvironment and are closely associated with tumorigenesis at all stages, including DNA damage, immune surveillance evasion, and microbial synergistic effects. Microorganisms contribute to carcinogenesis through interactions with host immune systems and signaling pathways, leading to immune activation and cellular proliferation. As the primary source of nutrients and energy, diet plays a vital role in sustaining life, and the role of dietary components in chronic inflammation development is increasingly recognized. As a hotspot in nutritional epidemiology, various dietary patterns and inflammation-based scoring systems have been developed to investigate diet-disease relationships, including the Mediterranean Diet Adherence Score (MEDI-LITE), Healthy Eating

Index (HEI), and DII. Given the collinearity among dietary components, assessing overall dietary patterns better overcomes limitations of evaluating single components or nutrients. Based on global monitoring data, DII not only qualitatively distinguishes between anti-inflammatory and pro-inflammatory dietary tendencies but also quantitatively evaluates the total inflammatory potential of dietary patterns, with its validity confirmed by multiple serum inflammatory markers such as CRP, IL-6, and TNF-α.

This meta-analysis pooled 11 studies comprising 9,015 participants to evaluate the DII-UGIC relationship. The findings demonstrate a positive association between high DII scores and UGIC risk. In categorical analysis, individuals in the highest DII category had an 81% higher UGIC risk compared to the lowest category, with the magnitude varying by tumor type: gastric cancer risk increased by 95%, esophageal cancer by 120%, and esophageal squamous cell carcinoma by 168%—slightly higher than esophageal adenocarcinoma (159%). In continuous analysis, each one-unit DII increase conferred a 53% increase in UGIC risk. Subgroup analysis revealed a more pronounced risk increase in females than males. These findings underscore the potential benefits of anti-inflammatory diets for UGIC prevention and hold important public health implications.

Similar results have been reported in meta-analyses examining DII and other cancers: highest DII categories were associated with a 25% increase in breast cancer risk (RR = 1.25, 95% CI: 1.09–1.44), 73% increase in prostate cancer risk (OR = 1.73, 95% CI: 1.34–2.23), and 107% increase in head and neck cancer risk (OR = 2.07, 95% CI: 1.82–2.35). A study on DII and overall cancer risk demonstrated a 25% increase in cancer incidence (RR: 1.25, 95% CI: 1.16–1.35) and 67% increase in cancer mortality (RR: 1.67, 95% CI: 1.13–2.48). Chronic inflammation more extensively participates in epithelial tumor development, with robust evidence linking pro-inflammatory diets to colorectal cancer. This study similarly demonstrates markedly elevated risks for gastric and esophageal cancers. The stronger association between inflammatory diets and gastrointestinal cancers may relate to complex immune regulation from inflammation-activated immune systems. Dietary components and nutrients directly contact the digestive tract, where chronic inflammation upregulates various cytokines and chemokines that stimulate progenitor cell recruitment and engraftment into gastric cancer tissues. Cytokines further recruit and activate inflammatory cells such as neutrophils and macrophages, generating reactive oxygen species that impose oxidative stress on gastric epithelial cells and induce carcinogenesis. Similarly, the esophageal tumor microenvironment is mediated by cytokine levels, with inflammatory mediators such as vascular endothelial growth factor, CRP, and IL-8 inducing neovascularization and inhibiting immune cell recruitment to tumor sites. Inflammation also modifies the extracellular matrix, providing structural support for tumor growth.

Diet represents a series of complex interacting exposures with cumulative effects on inflammation and carcinogenesis. However, as a modifiable personal factor, developing rational dietary strategies is particularly important, especially

among high-risk populations. Anti-inflammatory diets may thus constitute an important measure for reducing cancer risk. The DII scoring system provides an effective tool for quantifying dietary inflammation and offers valuable evidence for cancer etiology and prevention strategy development.

Although all included studies were of moderate-to-high quality, with control groups matched for age and sex and comprehensive covariate adjustment, several limitations exist. All studies were case-control designs, limiting causal inference compared to prospective intervention trials. While uniform use of FFQ for dietary assessment reduced heterogeneity and enhanced comparability, implementation variations existed: three studies used self-administered FFQs and three used interviewer-administered FFQs. Self-administered FFQs may involve dietary recall avoidance and social desirability bias. Additionally, FFQs retrospectively collect dietary intake frequency and quantity over extended periods, introducing inevitable recall bias. Dietary habits may also change during follow-up periods. Furthermore, the included studies comprised only European and Asian data; given substantial dietary variations across populations, regions, and cultural backgrounds, validation in larger-scale, more geographically representative prospective studies is needed.

In summary, compared with low pro-inflammatory diets, high pro-inflammatory diets may increase UGIC risk, with more pronounced increases observed in female populations. Under professional nutritional guidance, reducing pro-inflammatory dietary intake is advisable. These conclusions require validation in larger-scale, prospective, multicenter clinical trials. As a novel tool for assessing dietary inflammatory potential, DII warrants further refinement. Future research should clarify the complex relationships among diet, inflammatory markers, and related diseases, explore underlying biological mechanisms, and provide additional insights for personalized cancer prevention and therapeutic targets.

Author Contributions

Zhai Leilei contributed to methodology development, study implementation, review, and writing. Zhai Leilei and Zhao Shupeng conducted literature screening and data extraction. Zhao Shupeng performed data analysis and visualization. Yao Ping provided research planning, guidance, quality control, and funding support.

Conflict of Interest

All authors declare no conflicts of interest.

Data Availability Statement

The scientific data supporting this study have been publicly released in the Science Data Bank of the Chinese Academy of Sciences and can be accessed at https://doi.org/10.57760/sciencedb.06954 or https://cstr.cn/31253.11.sciencedb.06954.

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